organelle continuous with the NE, and provides a platform to direct NE-recruitment of ESCRT-III during mitotic exit. CHMP7's N-terminus comprises tandem Winged-Helix domains and by using homology modelling and structure-function analysis, we identify point mutations that disrupt membrane-binding and prevent both ER-localisation of CHMP7 and its subsequent enrichment at the reforming NE. These mutations also prevent assembly of downstream ESCRT-III components at the reforming NE and proper establishment of post-mitotic nucleo-cytoplasmic compartmentalisation. These data identify a novel membrane-binding activity within an ESCRT-III subunit that is essential for post-mitotic nuclear regeneration.

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Lem2p (LEM2) and Cmp7p (CHMP7) function in ESCRT-dependent nuclear envelope remodeling in fission yeast.

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ESCRT-III proteins have been implicated in sealing the nuclear envelope in mammals, both during nuclear assembly and following mechanical disruption. This sealing process requires the ESCRT-II/ESCRT-III hybrid protein CHMP7 and the AAA ATPase VPS4. It remains unclear, however, how CHMP7 is recruited to breaches of the nuclear envelope. The fission yeast *S. pombe* is an attractive genetic model system for investigating this role of the ESCRT pathway because, in fission yeast, the nuclear envelope develops fenestrations that must be closed twice per cell cycle: upon mitotic entry when duplicated spindle pole bodies (SPB) are incorporated into the nuclear envelope and after a successful cell cycle when the SPBs are ejected back to cytoplasm. Here we report that deletion of fission yeast *vps4* leads to severe defects in nuclear morphology and integrity, which causes delayed segregation of duplicated SPBs, asymmetric nuclear bipartition in mitosis, and slow growth. Interestingly, these phenotypes are spontaneously suppressed by loss-of-function mutations that arise in *cmp7* (*pombe* CHMP7) or *lem2*, a member of the LEM (Lap2-Emerin-Man1) family of inner nuclear membrane proteins—implying that all three function in the same pathway. Based on these observations, we hypothesize that Lem2p acts as a nuclear site-specific adaptor to recruit Cmp7p to the nuclear envelope.